A DIFFICULTY IN PARTURITION.

By J. DAVIDSON, M.B. LOND., &c.

MRS. M—— was confined of her second child on Dec. 16th, 1888. Labour began at 3 A.M., and I was sent for at 11 A.M. The pains were strong, and the os was well dilated, but the head would not come down, so I delivered her with forceps at about 1.45 P.M. Great difficulty was experienced in delivering the shoulders. The cord was round the neck of the child once, and was very tight. Traction applied to the axilla produced very little effect, and simultaneously with each effort at traction the child's face became livid, resuming its natural colour again during the intervals between traction. As the cord was too tight to pass over the head or shoulders, I tied it in two places where it was round the neck, and divided it, when the shoulders were immediately born with the greatest ease. The child's heart was beating well, but respiratory movements were absent; these, however, were well established after two or three minutes. The cord was unfortunately not measured, but its length was about twelve or at the most fourteen inches from placenta to umbilicus. I examined the patient almost immediately after delivery, and found the placenta in the vagina.

Where there is every prospect that delivery of the child will immediately follow division of the cord in circumstances similar to the above, this would appear to be a much safer mode of treatment than waiting till the child and the placenta are both expelled together.

Uxbridge.

CASE OF RUPTURE OF THE HEART.

By JOHN HENRY ASHTON, M.R.C.S. ENG.

THE son of Mrs. S—— sent for me on Sunday morning, Nov. 12th, to see his mother. Death, however, had occurred before my arrival. The coroner, Mr. Braxton Hicks, directed me to make a post-mortem examination. The deceased, I learned, was seventy-three years of age. I found the body well nourished. On opening the thorax, the pericardium was distended by fluid and clotted blood; on removing this, a rupture, three-quarters of an inch long, could be seen on the anterior surface of the left ventricle: this extended through its walls into the cavity, and from it blood could be made to flow by pressure, the cavity of the ventricle being full. The right ventricle was empty, and the tricuspid orifice slightly dilated; the valves were healthy. The mitral valves were somewhat thickened. The heart was not enlarged; the muscular substance was mottled in colour, softened, and very easily lacerated; the fibres, examined under the microscope, had undergone fatty degeneration. A large gall stone almost entirely filled the gall bladder, and a small hard tumour was found at the lower border of the omentum adherent to the peritoneum and intestines.

The following history of the case was given me. The general health of the deceased was very good. Three years ago she had an attack of syncope, which lasted one hour and was attended by partial unconsciousness; since then she has been well, with the exception of experiencing shortness of breath when she has had to go upstairs. She had recently an attack of jaundice, which quickly passed away, and since then she has complained of cold feet. She has never suffered from pain in the side, or sickness. On the morning of her death she was about her usual duties, when she suddenly fell down, and died almost immediately.

Remarks.—The abiding shortness of breath upon exertion, with the prolonged attack of syncope, would have sufficed during life to have called attention to the state of the heart. It is interesting to note that the large gall stone gave rise to but trifling, if any, symptoms. The place of rupture was observed to be where the evidences of degeneration were most marked, and, as in the majority of recorded cases, took place in the left ventricle and on its anterior surface; the direction of the rent was in the course of the fibres, and about one-third of the distance from the apex to the base.

Sheen Park, Richmond.

A Mirror OF HOSPITAL PRACTICE, BRITISH AND FOREIGN.

Nulla autem est alia pro certo noscendi via, nisi quamplurimas et morborum et dissectionum historias, tum aliorum tum proprias collectas habere, et inter se comparare.—MORGAGNI *De Sed. et Caus. Morb.*, lib. iv. Proœmium.

ST. MARY'S HOSPITAL.

A CASE OF PERNICIOUS ANÆMIA, WITH RECOVERY.

(Under the care of Dr. BROADBENT.)

MUCH has been written on the subject of "pernicious anæmia" during the years which have elapsed since cases of this kind were first described and differentiated by Addison in his lectures at Guy's in 1843,[1] but there still remains some difficulty in distinguishing them from the anæmia found to accompany malignant and some other diseases of obscure character. Dr. Wilks recorded cases in 1847-8 (Guy's Hospital Reports). Addison appears to have called it "idiopathic anæmia," but the disease was not generally recognised as a clinical entity until the year 1872, when Biermer of Zurich wrote about it under the name of "progressive pernicious" anæmia, and awakened the attention of the profession to its characteristics and fatal termination. We should hardly think it necessary to refer to these points in the history of the disease, which are generally conceded, were it not that the credit appears to have been given to Biermer as the first discoverer in a recent review of a paper by Trechsel.[2] The literature on the subject is now voluminous, and we are not able to do more than refer to it, and mention some of the observers who have made it a special study. In 1877 Dr. Fenwick dwelt in his lectures on the occurrence of a similar condition of the system in cases of gastric atrophy.[3] In 1878 the subject was discussed at the Pathological Society,[4] and later in that year[5] Dr. Stephen Mackenzie, in a clinical lecture, referred to the history of the disease, and to an able review by Dr. Pye-Smith of a memoir by Lepine,[6] and the work of recent writers. Dr. Sidney Coupland, in his Gulstonian Lectures in 1881,[7] discussed fully both the disease and its treatment. Dr. William Hunter last year recorded his investigations into this disease; and these offer not only great additions to our knowledge on the subject, but are most important in elucidation of its pathology.[8] We commented[9] on his conclusions, and indicated the chief deductions to be drawn from his investigations at the time. 1. That the disease does not depend upon any impairment of the normal blood formation. 2. The excessive quantity of iron which he found in the liver cells (confirming the results obtained by Quincke and others) is due to an excess of the normal blood disintegration in that organ. 3. The disease depends upon the entrance of some poison, possibly of a cadaveric nature, generated in the gastro-intestinal tract, which gains entrance into the portal system and causes the blood to undergo disintegration therein. The principal pathological changes beyond those mentioned are those of fatty degeneration of the internal organs. We cannot enter upon the question as to the symptoms which distinguish the disease from other forms of anæmia; they are well shown in the case which we publish and are set forth in the papers to which we have referred, also in cases recorded by Immerman of Basle,[10] Quincke,[11] Bramwell,[12] Finney,[13] Padley;[14] and we have published cases in the "Mirror"[15] illustrative of it. Until the publication of Dr. Byrom Bramwell's paper on the

1 New Sydenham Society. Writings of Thos. Addison, p. 212.
2 The Etiology of Pernicious Anæmia. Revue Médicale de la Suisse Romande, 1888, in the Medical Recorder.
3 THE LANCET, vol. ii., pp. 1, 39, 77.
4 Ibid., vol. i., p. 495. 5 Ibid., vol. ii., p. 799.
6 London Medical Record, 1877. 7 THE LANCET, vol. i., p. 531, &c.
8 Ibid., vol. ii., p. 555. 9 Ibid., vol. ii., p. 678.
10 British and Foreign Med. Chir. Rev., Oct. 1874.
11 Med. Times and Gaz., 1876. 12 Ibid., 1877.
13 Brit. Med. Journal, 1881. 14 THE LANCET, 1883, p. 811.
15 Ibid., vol. i. 1878, p. 13, S. Mackenzie; vol. i. 1881, p. 13, Mitchinson; vol. i. 1883, Carrington.

success obtained by the use of arsenic in pernicious anæmia, it was looked upon as truly progressive, and always to a fatal termination. Since that time there has been a change, and many cases have been published proving the efficacy of arsenic, recovery having frequently followed its employment; sometimes there appears to have been a relapse, and sometimes it has failed, but no treatment has yielded such good results. Iron, which in cases of ordinary anæmia is so efficacious, appears to be almost useless, but a case is recorded in which arsenic failed to cure and the patient recovered on the administration of iron. Other treatment adapted to improve the general health, allay digestive disturbance, produce appetite, &c., has been recommended; and transfusion has been tried in some instances with success (see Coupland), but the use of arsenic is generally indicated. We dwell on this point, as a recent edition of a work on therapeutics does not draw attention to its great properties in such cases. Dr. Broadbent, in a former case[16] which recovered, that of a woman, aged forty-three, who had been suffering for four months, gave the drug in frequent small doses, on the failure of iron, and obtained a cure in two months; in this case he followed a similar plan. It is unusual to find enlargement of the spleen in these cases. Henrot found it enlarged post mortem in two cases, and considers that the state of this organ has much to do with the disease; this view is, however, negatived by the investigations of Dr. Hunter. For the following report we are indebted to Mr. J. J. Clarke, M.B. Lond., late house physician.

E. V——, a well-developed woman aged twenty-six, was admitted on Feb. 7th, 1888, complaining of headache and vomiting. For seven years she had worked as a domestic servant, and for the last year as a cook. She was in the habit of taking one pint of beer a day. She says she has always been subject to "headache and biliousness." She dated her illness from Christmas last, since when she had lost appetite and flesh, and had had frequent headache and sleeplessness. A fortnight ago her friends first noticed the yellowness of her skin. She began to vomit eight days before admission, and has vomited at intervals ever since. The catamenia have been regular up to the day of admission. When first examined the patient looked very anæmic and jaundiced, but the former condition was the more marked of the two. The staining of the skin was universal, but the sclerotics were unaffected. The fat at the canthi was stained yellow. The lips and mucous membrane were slate-coloured. There was no marked wasting. The vessels of the neck throbbed violently. The patient was very restless; she kept her eyes closed, and was continually moaning. She complained chiefly of headache. She had tinnitus, and she could not sleep. The tongue was thickly coated with a moist white fur. There was great anorexia, pain after food, and vomiting of bile-stained fluid. The spleen was enlarged, reaching nearly to the middle line across the upper part of the epigastrium. It was firm and tender. The stools were soft and of a normal colour. The radial arteries were small, and not felt between the beats. The wave was small, short, and easily arrested. The rate was 114 and regular. The apex beat was seen and felt just outside the nipple line in the fifth space. There were soft blowing murmurs over the apex and the aortic and pulmonary cartilages, and a marked venous hum was heard in the neck. Under the microscope, the blood was seen to contain much fine granular matter and irregular granular bodies, about eight times the size of a leucocyte. The red corpuscles were greatly reduced in number, an average of seven only being counted to the square of Gowers' slide, instead of fifty-four. Many of the corpuscles were tailed or otherwise deformed. On examining the eyes, the pupils were seen to be unequal; the left was the larger, and on this side the cornea was found to be conical. The outlines of both discs were hazy, but there was no distinct papillitis. Both retinæ showed extensive recent hæmorrhages and also white patches. On the left side the yellow spot was found to be involved in a large hæmorrhage. The urine was concentrated, and deposited uric acid crystals and urates on standing. The temperature on admission was 102°. The patient was put on liquid food, administered frequently. A sixth of a grain of morphia with a twentieth of a grain of hyoscyamine were injected under the skin at night.

On Feb. 10th Dr. Broadbent saw the patient, and ordered her two minims and a half of liquor arsenicalis every three hours. The next day there was a marked diminution in the tenderness of the spleen, and the vomiting ceased, though the pyrexia and other symptoms were still severe. The evening temperature on the 11th and 12th was 104°; the highest point reached afterwards was 102·5.° There were never any signs of arsenical irritation. The improvement was gradual but decided. On the 21st a tinge of colour could be seen in the lips and cheeks, and the evening temperature reached only 100·5.° As the temperature fell the pulse rate increased and was now 144. The headache was much less, and the tenderness of the spleen had gone altogether. From the time arsenic had been administered vomiting occurred on an average once in forty-eight hours. After this day the improvement went on more rapidly, so that on Feb. 28th the temperature had fallen to normal and the pulse rate to 100. The headache and vomiting had ceased for some days. The heart was still dilated; the same murmurs were present. The spleen was very slightly smaller than on admission, and not at all tender. The sight had improved, and the hæmorrhages into the retina were rapidly clearing up. The improvement went on steadily, so that on March 15th the patient expressed herself as feeling perfectly well. The patient was allowed to sit up for a short time on March 21st and succeeding days, but on the 28th swelling of the feet, headache, and nausea were present, and the patient was again confined to bed. On May 15th she got up again, and on the 20th left the hospital for a convalescent home. At that time the anæmia was less marked and the tongue clean and red. The cardiac murmurs were absent, and the spleen, though still large, was not tender. The catamenia were absent all the time she was in the hospital. Two months afterwards she returned to show herself. She was feeling well; there was a good deal of colour in the lips and cheeks, and the catamenia had returned.

Remarks.—The clinical phenomena are distinctive of pernicious anæmia: a lemon-yellow skin with sclerotics unaffected, and a temperature reaching 104°, are the leading features. The marked effect of arsenic and the tolerance of it are also characteristic. There was no history of catamenial trouble dating from puberty, as there would probably have been had the condition been an extreme degree of chlorosis. The patient's occupation (cook) points to the alimentary canal as the seat of the determining lesion.

[16] Brit. Med. Jour., 1880, p. 503. See also Wilks on "The Therapeutic Value of Arsenic in Anæmic Cases," THE LANCET, vol. i. 1885, p. 653; Withers Moore, British Medical Journal, vol. i. 1881, and Tyrrell Brooks, THE LANCET, vol. ii. 1887, p. 849; and Copeman "On the Changes of the Blood," ibid., vol. i. 1887, p. 1076.

LEEDS GENERAL INFIRMARY.

TWO CASES OF SUPPURATIVE SYNOVITIS, WITH COMPLETE RECOVERY OF THE JOINT.

(Under the care of Mr. A. W. MAYO ROBSON.)

IN these cases of suppuration in the knee joint most satisfactory results were obtained by the treatment adopted, the patients recovering with perfectly movable joints. In neither case, however, was there decomposition of the pus, and in the one which was afterwards treated by incision the part was kept in a condition of asepsis by dressings, which proved sufficient. We are inclined to think the remarks of Mr. Treves on his case refer more to a septic condition of the joint than to such instances as these, for in such it is very rare to obtain anything like satisfactory movement after any method of treatment, and the surgeon is frequently thankful to save the limb even with a stiff joint. In the following record the line of treatment is shown for acute suppurative synovitis: aspiration, followed by incision and efficient drainage, when necessary from repeated accumulation of pus, extension beyond the limits of the joint, the severity of the local or general symptoms. The notes of the cases have been furnished by Mr. Berkeley G. A. Moynihan, M.B., house surgeon.

CASE 1.—Charlotte B——, single, aged twenty-six, was admitted into the infirmary on March 23rd, 1885, with the following history. Whilst going downstairs a few days previously, she felt her knee give way beneath her, but she did not fall; she was unable to straighten the limb, and in about two hours noticed that the knee joint was swollen.